

Hepatic Ischemia, Caused by Celiac Axis Compression, Complicating Pancreaticoduodenectomy

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Objective

In the course of pancreaticoduodenectomy, profound hepatic ischemia developed in two patients (one with ampullary carcinoma, the other with chronic pancreatitis). This article addresses the diagnosis and correction of the celiac axis compression responsible in this complication.

Summary Background Data

Since hepatic ischemia appeared immediately after division of the gastroduodenal—pancreaticoduodenal arcade, which provides mesenteric to celiac collateral circulation, celiac axis narrowing or occlusion was suspected. Previous reports have indicated that celiac axis disease may be present in about 10% of such patients.

Methods

Doppler flow studies, and in the second patient, intraoperative angiography were performed. The celiac axis was exposed and mobilized in both.

Results

Initially, no flow could be detected in the celiac axis. Dense fibrous tissue was found encasing it. Division of the entrapping tissue restored flow to the upper abdominal viscera.

Conclusions

The anatomic deformation of the celiac axis predisposing to this complication is detectable on the lateral projection of a preoperative celiac angiogram. If, however, an angiogram has not been done, an initial test occlusion of the gastroduodenal artery before its division permits anticipation of the complication, correction of the celiac impingement, and hence, avoidance of hepatic ischemia.

A decade ago, Thompson and associates¹ reported in this journal two patients with celiac axis stenosis or oc-

clusion who required revascularization of the celiac circulation during pancreaticoduodenectomy. In both instances, the atherosclerotic lesions had been identified preoperatively by selective visceral angiography.

Recently, Kohler et al.² described a third patient who underwent pancreaticoduodenectomy and likewise had compromise of the celiac circulation. In that patient,

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however, the etiology was axis compression by the median arcuate ligament; this was successfully corrected by dividing the impinging diaphragmatic fibers, releasing the celiac artery.

We report two additional patients undergoing pancreaticoduodenectomy in whom obstruction of the celiac arterial circulation was not suspected until pancreatic resection had been achieved. Then, obvious ischemia of the liver, stomach, and pancreatic remnant led to discovery of celiac arterial occlusion, caused in both patients by compression and/or kinking of the axis by dense fibrotic tissue about its origin. Division of this peri-aortic tissue relieved the obstruction, successfully restoring blood flow to the upper abdominal viscera.

Case 1

RC, a 67-year-old white man, presented with a 2-month history of progressive anorexia, fatigue, jaundice, acholic stools, and weight loss. A preoperative evaluation, including abdominal ultrasonography, abdominal CT scan, and endoscopic retrograde pancreatography, identified dilated intrahepatic and extrahepatic ducts and a 1.5-cm filling defect in the distal common bile duct at the ampulla. The latter was biopsied endoscopically and found to be adenocarcinoma. Visceral angiography was done preoperatively. On the standard anteroposterior projection, the origin of the celiac axis appeared dilated; the common hepatic artery was normal in appearance and there was a prominent gastroduodenal artery (Fig. 1). When the superior mesenteric artery was injected, a prominent pancreaticoduodenal arcade was identified; there was retrograde filling of the gastroduodenal and the proper hepatic arteries (Fig. 2).

On June 15, 1990, the patient underwent exploration. No evidence of metastatic disease was found. Consequently, the duodenum, head of the pancreas and distal third of the stomach were resected en-bloc. However, after reconstruction with pancreaticojejunostomy and choledochojejunostomy, the liver and stomach were noted to be pale and obviously ischemic. With Doppler examination of the common hepatic artery, minimal flow was detected. There was no palpable pulse in the celiac axis; extensive fibrosis was found about its origin. This was completely lysed, after which a strong pulse was palpable in the celiac axis and common hepatic artery. The liver and stomach promptly resumed their normal appearance. The gastrojejunostomy was then completed and the patient recovered uneventfully. The following day the patient underwent selective visceral arteriography, which confirmed patency of the celiac axis and normal flow in the hepatic artery and its branches (Fig. 3). The patient's plasma AST and ALT concentrations reached maximal values of 1056 U/mL and 1020 U/mL, respectively, on the first postoperative day.

Case 2

GW, a 45 year-old white man, had a 10-year history of chronic pancreatitis characterized by deteriorating exo-



Figure 1. The preoperative contrast injection of the celiac axis in patient R.C., demonstrating post-stenotic dilatation of the celiac artery, a normal hepatic artery and a prominent gastroduodenal artery.

crine and endocrine function and incapacitating abdominal pain. An ERCP demonstrated multiple strictures of the pancreatic duct within the head of the gland and a distal common bile duct stricture. On December 23, 1991, the patient underwent laparotomy. At exploration, which included intraoperative ultrasonography and contrast pancreatography, multiple calcifications were identified throughout the pancreatic duct. There was marked scarring with multiple small diameter channels present throughout the head of the pancreas. The distal duct was dilated. Because of the extensive disease in the head and adjacent body of the pancreas, it was decided to proceed with pancreaticoduodenectomy, preserving the tail of the pancreas to retain some endocrine function. The pancreaticoduodenal complex was mobilized, after which the gastroduodenal artery was sacrificed. About 5 to 10 minutes later, the liver was noted to be diffusely dusky. Only a weak pulse was palpable in the common hepatic artery and celiac axis. Doppler flow was barely detectable in those vessels. The hepatic artery was cannulated but no arterial wave form was detected. A retrograde hepatic arteriogram (Fig. 4) showed an entirely normal but small hepatic and splenic arterial system, without obstruction or thrombosis. However, there was no retrograde flow via the celiac axis into the aorta.



Figure 2. Preoperative contrast injection of the superior mesenteric artery in patient R.C. A prominent pancreaticoduodenal arcade fills in retrograde fashion the gastroduodenal artery and then the proper hepatic artery.

Upon dissection about the celiac axis, dense fibrosis was encountered. This was sharply lysed, relieving a pronounced kink of the celiac axis and promptly restoring flow through it. A strong pulse and excellent Doppler flow were easily identified in the celiac, splenic and hepatic arteries. The pancreaticojejunostomy, choledochojejunostomy, and gastrojejunostomy (it was decided not to perform a pylorus preserving procedure) were then completed.

Postoperatively, maximal AST and ALT concentrations of 822 U/mL and 792 U/mL were reported on the second postoperative day. He otherwise had an uneventful postoperative recovery. Four months later, however, he presented with an acute abdominal catastrophe and died within hours of admission. At autopsy, a perforation of the jejunum was found with no evidence of disease involving the mesenteric vessels.

DISCUSSION

Although both Thompson et al.¹ and Kohler et al.² suggested that celiac artery stenosis or occlusion might predispose to morbidity or mortality after pancreaticoduodenectomy if not discovered and corrected, in the

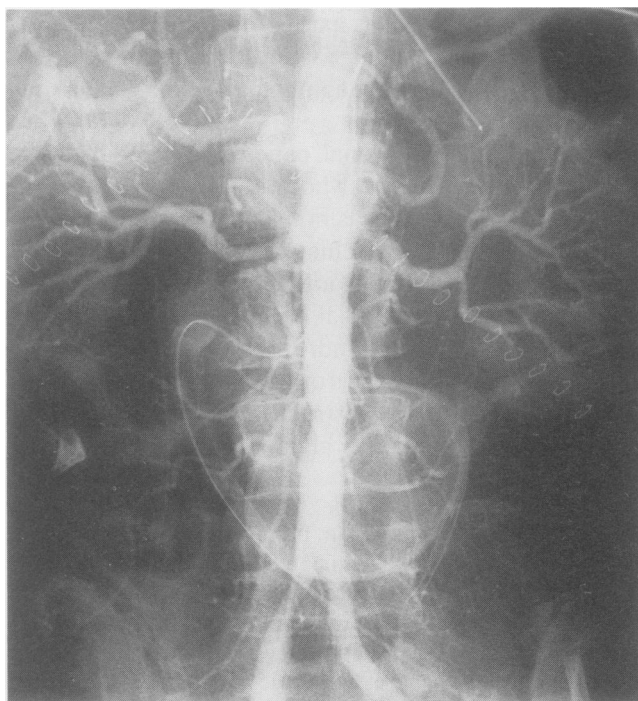


Figure 3. Postoperative angiography in patient R.C., demonstrating a patent celiac axis with normal filling of the hepatic artery and its branches.

three cases reported by them the lesions had been identified preoperatively and were bypassed before interrupting the pancreaticoduodenal arcades. Consequently, the upper abdominal viscera were not rendered ischemic intraoperatively.

In contradistinction, the diagnosis of celiac axis occlusion was made in our patients only after the liver, stom-

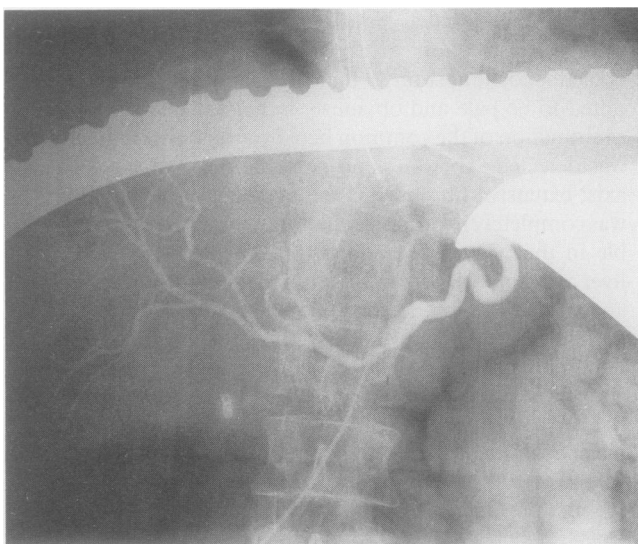


Figure 4. Intraoperative retrograde hepatic angiography in patient G.W., during the interval of acute hepatic ischemia. The hepatic and splenic arteries are readily visualized but there is no retrograde filling of the aorta.

ach, and pancreatic remnant became profoundly ischemic. In both instances, a strong pulse had been palpated in the hepatic artery before pancreatic resection and concomitant ligation of the gastroduodenal artery. After removal of the specimen, the pulse in the hepatic artery had disappeared. Only then was the dense fibrotic tissue ensnaring and kinking the celiac axis discovered and subsequently divided. This released the axis, with prompt and complete resolution of the circulatory impairment.

Peripancreatic inflammation in response either to ductal obstruction from ampullary carcinoma (case 1) or especially from chronic pancreatitis (case 2) may have caused or contributed to the celiac entrapment. Thompson et al. emphasized that celiac axis stenosis or occlusion is not a rare finding; they documented an incidence of 10.5% in 200 selective visceral angiograms.¹ Most commonly the lesions were atherosclerotic and, consequently, when requiring correction are best treated by creating a bypass linking the aorta or superior mesenteric artery to the celiac system. Their preference, in the setting of pancreaticoduodenectomy, was splenic artery to superior mesenteric artery implantation.¹

Thompson and associates¹ espoused routine selective visceral angiography before possible pancreaticoduodenectomy. Although modern imaging techniques (CT, CT portography, and MRI) have largely supplanted angiography for the evaluation of resectability, detection of hepatic metastases and differentiating neoplastic from inflammatory lesions, preoperative visceral angiography

is of inestimable value for mapping the midgut arterial anatomy, which is prone to frequent anomalies and variations. Our experience and that previously reported^{1,2} suggest that a lateral projection of the celiac axis (and superior mesenteric artery) should be an integral part of the study to detect either arteriosclerotic narrowing and occlusion or extrinsic compression and kinking.

If the appropriate angiographic studies have not been obtained before pancreaticoduodenectomy, we recommend that a test occlusion of the gastroduodenal artery precede its ligation. The hepatic arteries are palpated before and during the test occlusion. If the pulse diminishes during occlusion or if there is evidence of upper abdominal visceral ischemia, impairment of flow should be verified by Doppler examination. If celiac obstruction is confirmed, the celiac axis is exposed and examined for intrinsic lesions or extrinsic compression and the appropriate remedy implemented.

In our two patients, failure to identify and hence to correct the celiac arterial insufficiency occurring after the obligatory interruption of collateral arcades in the course of pancreaticoduodenectomy would likely have resulted in serious morbidity if not mortality.

References

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